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Non-epileptic attack disorder: a psychological perspective

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Due to the serious medical consequences in failing to recognize non-epileptic attack disorder (NEAD), and the frequency with which neurologists come into contact with such patients, clearly NEAD constitutes a major concern for clinicians in the field of epilepsy. This article presents the psychological characteristics of 185 patients with NEAD. Psychological factors that were identified as being important in the understanding of the development and maintenance of NEAD included: anxiety or stress; physical abuse; significant bereavement; family dysfunctioning; relationship problems; depression; sexual abuse. An absence of relevant psychological factors was found in only 5% of patients. From patients' descriptions of their attacks, it appears that many symptoms are related to anxiety. Our findings are largely supported by previous studies and their relevance to effective management and treatment of NEAD patients is discussed.

Key words: Non-epileptic attacks; psychological factors.

INTRODUCTION

The importance of the early diagnosis of non-epileptic attack disorder (NEAD)[1] has been well recognized, as failure to do so can lead to certain risks for the patient: polypharmacy and toxicity[2]; hazardous interventions[3] including repeated venous cut-downs[4]; social and economic demands[1]; neglect of underlying psychological distress[5]. It is beneficial for a rapid diagnosis to be made; clinicians have noted that those NEAD patients diagnosed and treated earlier have a better prognosis[3,6,7].

Whilst there has been extensive research concerning the psychiatric correlates of NEAD, psychological studies have been largely limited to Minnesota Multiphasic Personality Inventory (MMPI) profiles. As there is an absence of clear correlations between NEAD and hysterical personality traits[5,8,9] or a reliable MMPI profile[10–13], it has been suggested that clinicians and researchers move away from a personality-driven, psychodynamic view of NEAD and adopt a behavioural explanation for the disorder[9]. As such, it appears that NEAD consists of a learned pattern of behaviour which is developed to enable the person to deal with extreme stressors, either internal or external. Viewing such seizures as learned behaviour is supported by observations that such attacks do not often appear in people who have no experience of epilepsy, i.e. a behavioural model is usually required[14,15]. Although the seizure is thought to begin as an anxiety-reducing mechanism[9], thus constituting the primary gain, environmental factors soon confer secondary gains, such as evasion of responsibility and manipulation of people and circumstances. These gains presumably act as powerful reinforcers serving to perpetuate the behavioural pattern.

Clinicians have consequently recognized the importance of assessing psychological factors in people with such attacks[10,16–18]. Unfortunately, this area has been approached primarily with a view to simplifying the process of differential diagnosis[16,19,20]. Roy[19] identified five differentiating factors, those with NEAD scoring higher on each of the following: family history of psychiatric disorder; past personal history of psychiatric disorder; attempted suicide; current affective syndrome; sexual maladjustment. These factors are consistent with those found in subsequent studies[3,5,21–23]. Other psychological factors found to be related to NEAD include family dysfunctioning[10,24], marital disharmony[14,25,26], childhood sexual abuse[27–34], bereavement[35], depression[22,36,37] and anxiety[19,26].

In this study we examine the psychological factors associated with a diagnosis of NEAD in 185 patients

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Table 1: Information collected by pro-forma for each patient with NEAD.

Category	Information collected
Demographic information	Age, gender, marital status, occupation, area of residence, whether in receipt of state benefits.
Referral information	Referred by whom, date referred, description of problem, prior diagnosis of epilepsy, whether referred previously with NEAD.
Medical details	Prescribed medication, medical history of note, known relatives with diagnosis of epilepsy, learning disability.
Psychological details	Significant family dynamics, bereavements, relationship difficulties, separation/divorce and other life stresses, sexual/physical abuse, evidence of mood disorders, suicide attempts.
Previous therapeutic input	Which services involved in providing therapeutic input in the past, reason for input and what was provided.
Information concerning attacks	Investigations carried out and results, reported symptoms, age at onset of attacks.
Clinic data	Appointment with psychologist, number of contacts, date of report, which psychologist seen, recommendations of report.

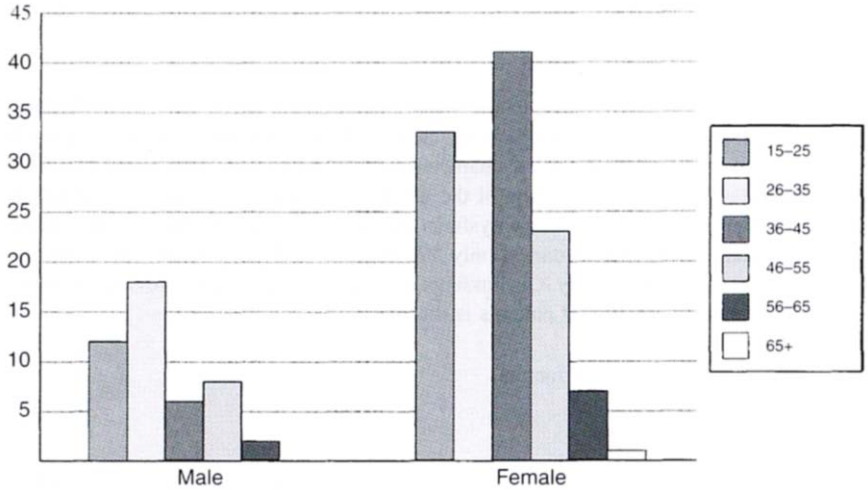

Fig. 1: Numbers of males and females in each age range as listed.

who were referred to the Neuropsychology Department of the Walton Centre for Neurology and Neurosurgery over a 2-year period. It is proposed that this study adds significantly to the current literature due to the extremely large number of subjects. The aims of the study was to identify those psychological factors which are likely to play an important role in the development and maintenance of NEAD and therefore point towards effective means of treatment.

METHODS

A retrospective study was undertaken of all patients referred for psychological assessment of NEAD from January 1992 until March 1995. In total there were 185 patients. Case records were analysed using a *pro-forma* devised by the authors. In this article we will only examine the information pertaining to the psychological characteristics of this sample of patients. Table 1 details the manner of information collected for each patient.

RESULTS

Demographic information

There were 185 patients, of which 48 were male and 137 female (74%). The ages of patients ranged from 15 to 65, with a mean age of 35.6 years. The mean age of onset of attacks was 25.7 years, with the majority occurring between the ages of 10 and 20.

In terms of marital status, 44% were married, 31% single and 23% were divorced. Only 21% were in full-time employment with 62% unemployed and the remainder either in full-time education or retired. There were 37% of patients receiving disability or other state benefits. A previous diagnosis of epilepsy had been made in 52% and 73% were currently taking anti-epileptic medication. Only 20% had documented and definitive evidence of epilepsy; they were consequently diagnosed as having both epilepsy and NEAD. Knowledge of a friend or family member with epilepsy was held by 48% of patients.

Table 2: Description of symptoms during attacks ($N = 145$).

Symptoms described	Number ($N = 145$)	Percentage
Shaking/trembling/flailing arms, legs, whole body	49	34
Vacant/'staring'/unresponsive	31	22
Headache/migraine	29	20
'Blackout'	29	20
Dizziness/faint	26	18
Hyperventilation/difficulty breathing	25	17
Collapsing	24	17
Tingling sensation/pins and needles	17	12
Loss of consciousness/awareness	15	10
Aggression—throwing objects, self-injury, hitting others	13	9
Subjective feelings of fear/anxiety	11	8
Sweating/clammy	10	7
Confused/disorientated	9	6
Shouting/screaming	8	6
Visual/perceptual disturbances—flashing lights, blurred vision	8	6
Clear panic symptoms reported	8	6
Pains—in chest, neck, arms	7	5
Palpitations/rapid heart rate	5	4
'Butterflies in the stomach'	5	4
Palour	5	4
Nausea/vomiting	4	3
Obsessional/bizarre behaviour	4	3
Tongue biting/incontinence	3	2
'Buzzing' noise in the ears	3	2
Slurred speech	3	2
Crying/tearful	2	2
Arching back	2	2

Attack symptomatology

For each patient, descriptions of their attacks were obtained from one of three sources; the referral letter, self-report by client or report from a relative or witness. The most common symptom reported (34%) was shaking of some kind, which ranged from gentle trembling of a hand to violent flailing of all four limbs. During an attack, the patients were usually described as being unresponsive or 'vacant' (20%), some experiencing a 'blackout' (20%) or loss of awareness (10%). Many also report headache, 'tingling sensations', feeling dizzy and difficulty in breathing. Closer examination reveals that most of the symptoms could in fact be described as anxiety related, and some very clearly are (hyperventilation, subjective fear emotions, panic attacks) (see Table 2). Presumably, most attacks in our sample simulate tonic–clonic seizures, but there are also some complex-partial seizure symptoms, typical of temporal lobe epilepsy: *déjà-vu*; nausea; bizarre behaviour; perceptual disturbances; 'staring'. These were evident to a lesser extent in the patients' descriptions of their attacks.

Table 3: Psychological factors revealed through psychological interview with clients ($N = 177$).

Psychological factors	Number ($N = 177$)	Percentage
Anxiety, stress, breakdown	75	43
Physical abuse, assault	49	28
Significant bereavements	45	26
Relationship problems, family dysfunctioning	44	26
Depression	41	23
Disrupted childhood	28	16
Sexual abuse/rape	24	14
Pain, illness behaviour	19	11
Suicide attempts	17	10
Learning disabilities	15	9
Financial problems	13	8
Alcohol/drug problem	8	5
Eating disorder	5	3
Agoraphobia	5	3
Post natal depression	2	1
Post traumatic stress disorder	1	0.5
No psychological factors identified	8	5

Psychological characteristics

There were 23% of patients with a past psychiatric history, mainly consisting of treatment for anxiety, 'breakdown' and depression and 10% had attempted suicide. In addition, it was found that 38% had received some form of therapeutic input in the past. The majority received input from either a psychiatrist or a clinical psychologist. Other types of input were received from either a social worker or a specialist service, such as Relate, Alcoholics Anonymous, a medical pain clinic, counselling from the National Society for the Prevention of Cruelty to Children (NSPCC), or Ashworth Special Hospital (a regional forensic secure unit).

Psychological factors which were identified through the interview conducted by the clinical psychologist are listed in Table 3.

An absence of relevant psychological factors was found in only 5% of clients interviewed. Almost half had suffered, or were currently experiencing, anxiety disorders, including stress, panic attacks and 'breakdown'. Many of the attack symptoms, as previously described, could be reclassified as anxiety-related symptoms (see Table 2). After anxiety, the most common factors identified were physical abuse, significant bereavements, relationship problems/family tensions, depression and sexual abuse.

DISCUSSION

The preponderance of females with NEAD in our sample reflects this same finding in many other

studies[21–23,38]. Most patients began having attacks in their adolescent years, as opposed to the childhood onset more typically seen with epilepsy. Adolescence is recognized as a potential time of turmoil and psychological distress, when self esteem and important relationships are being developed and the person gains their own identity. It appears to be a time when predisposed individuals become vulnerable to non-epileptic attacks.

Although we are reluctant to stereotype patients with NEAD, if we were to describe the 'typical' patient in our sample, she would be aged 36 years with onset of attacks in adolescence, carrying a diagnosis of epilepsy and be taking anti-epileptic medication. She would be unemployed, single or divorced and know someone with epilepsy. A past psychiatric history may be present, with the patient having had treatment for anxiety from a psychiatrist or clinical psychologist. There will probably be reports of some significant negative life events, such as: physical abuse; bereavement; family and relationship problems; sexual abuse; attempted suicide. In addition the patient will probably show psychological distress in the forms of anxiety and depression.

Gates and colleagues[16] proposed an innovative nosology whereby psychogenic non-epileptic events are subdivided into consciously or unconsciously motivated attacks. Fenton[39] asserts that patients with NEAD are rarely aware of the underlying psychological motivation and the attacks should not be viewed as manifestations of 'faking'. From descriptions given by our patients of their attacks, it is clear that awareness is often not retained and therefore we would propose that the majority of our sample would fall into Gates' 'unconscious psychogenic' category. We have already observed that many of the symptoms described appear to be anxiety related[40]. For example, the following symptoms are identified by ICD-10[41] as characteristic of panic disorder and the majority of these are included in Table 2: 'abrupt onset; intense fear; palpitations; sweating; trembling or shaking; dry mouth; difficulty breathing; feeling of choking; chest pain or discomfort; nausea or abdominal discomfort; feeling dizzy, unsteady, faint or lightheaded; derealisation, depersonalisation; fear of losing control or passing out; fear of dying; hot flushes or cold chills; numbness or tingling sensations'. It is probable that for many of our sample their attacks are a further manifestation of high arousal levels and reflects a method used to contain anxiety. This proposal is further supported by the finding that most patients spontaneously reported problems with anxiety and, where they had a past psychiatric history, it included treatment for anxiety disorders in the majority of cases. It is pertinent that more than a third of our patients had previous contact with mental health services[20,42]. This raises the issue as to whether NEAD is manifest following chronic psychological distress which has not been amenable to routine psychotherapeutic treatment. Indeed, it is a widely held view amongst psychologists working with this population that NEAD originally develops as an anxiety-reducing mechanism[9], thus fulfilling a primary gain. It appears, however, that NEAD is not altogether successful in allowing the individual to avoid psychological distress, as anxiety and depression often co-exist with non-epileptic attacks[22,34,36,43].

Ramchandani and Schindler[35] make the observation that it is difficult to determine whether the symptoms of anxiety and depression are the cause or the effect of NEAD. It is likely that for some patients, NEAD may act as a dissociative state[34,35], causing the release of overwhelming emotions related to past traumatic events. The type of attack experienced may give some clue as to the precipitating event; Ramchandani and Schindler[35] suggest that NEAD patients displaying dissociative symptoms tend to experience complex partial-like attacks.

Some of the most important areas for assessment in the psychological interview when suspecting a diagnosis of NEAD are: the identification of psychological distress, the presence of other manifestations of psychological difficulties and significant negative life events that may have played a part in the aetiology and precipitation of attacks in a predisposed individual (i.e. the underlying primary gain being to avoid the distress produced). In order to achieve effective management and treatment, it is essential to identify any factors which serve to maintain the attacks (i.e. secondary gains) in the person's current life circumstances, such as financial incentives, avoidance of high expectations, responsibility or conflict, receiving care and attention from family and friends that would otherwise be lacking. Psychotherapy or family therapy may focus on the original negative event or trauma believed to have precipitated the attacks.

With the identification of significant psychological factors in 95% of our sample, and some of those factors being very serious (e.g. attempted suicide, sexual abuse, PTSD, severe periods of anxiety and depression which have required in-patient treatment), it is acknowledged that this group of people have led very difficult lives and that NEAD is possibly a physical manifestation of this. We found support for *three* of Roy's[19] five factors which he claims distinguishes people with genuine epilepsy from those with NEAD: past personal history of psychiatric disorder, attempted suicide and current affective syndrome. Information pertaining to the other two factors, namely family histories of psychiatric disorder and sexual maladjustments, was not collected in this study, although their presence can be extrapolated from the

findings of family, marital and relationship difficulties and sexual abuse.

The vast majority of our sample reported the presence of one or more psychological factors which have been reported previously by other researchers. Ramchandani and Schindler[35] found 'guilt-laden bereavement' to be an important precursor of attacks which clinically resembled complex partial seizures; Bowman[34] reports 70% of NEAD patients had experienced physical abuse. Our findings also fit in with the view of Roy[14,25] who postulates that the attack is often a signal of distress about a severe marital problem. Wilkus and colleagues[10] found that NEAD patients perceived more difficulties in early family relationships. Moore and colleagues[24] also found evidence that NEAD patients perceived their families as displaying less commitment and support than controls or people with epilepsy and they suggest a role for family involvement in therapy for NEAD. Scott[26] cites evidence that along with the accepted factors of depressive illness and suicide attempts, marital disharmony and family dysfunctioning implicate a diagnosis of NEAD. Other researchers have also found increased incidence of relationship problems[36] and conflict in family relationships[37]. In these situations, attacks may be a form of avoiding conflict and maintaining relationships. It is of note that 16% of our sample experienced a disrupted childhood, due to the absence of a mother or being in care. It may be hypothesized that in these clients, an attack may be an attempt to ensure care and attention, and guard against further abandonment from those who are currently significant in their life.

Of our sample 14% had been sexually abused or raped. Many other researchers have noted the frequency with which people with NEAD reveal that they have been sexually abused[27–34]. The prevalence of sexual abuse in any population is difficult to ascertain. The reliability of reported incidence and prevalence rates of sexual abuse has been hampered by the absence of a clear operational definition and inconsistencies in the methodology used to collect this information. Estimates of sexual abuse in people with NEAD range from a minimum of 10%[44] to 77%[34]. Gross[27] suggests that abused individuals use attacks as a 'defence mechanism' to escape the painful reality of their lives; it then becomes a learnt behaviour pattern, transferable to a variety of stressors outside the original context. Thus incest or sexual abuse may serve to precipitate the initial development of attacks in childhood, but need not be the underlying cause for later attacks. Bowman[34] suggests that later attacks are a means by which abused women 'dissociate' themselves from memories of early sexual abuse. It may be that people who have been abused tend to exhibit a particular type of non-epileptic attack which is identifiably distinct from attacks arising from other aetiologies. One of the ictal characteristics which is cited as predictive of sexual abuse is pelvic thrusting[45] and back arching[38], both of which were relatively absent in our patients' descriptions of their attacks. It has been found that, amongst child sexual-abuse victims, there is a higher life-time prevalence of medically unexplained physical symptoms and a greater use of health-care resources[46]. Pain or illness behaviour were exhibited by 11% of patients; these are considered to be other signals of underlying psychological distress. There were 9% of patients with learning disabilities; again this finding supports previous studies in the literature where Drake *et al*[38] found that 20% of their NEAD sample had learning disabilities.

Clearly, it would be of great benefit to examine the base rates of these experiences and adverse life events in the local population in order to see if our sample exhibits higher levels. This would then enable us to determine whether any of these factors are indeed correlated with NEAD or whether they are present in similar proportions in the local population who do not exhibit attacks. This is one suggested area of further work. We recognize that our present study would be enhanced by a prospective study investigating the long-term outcome of patients with NEAD. Clearly, the identification of psychological characteristics is only the first step towards the organization of specific and effective management and treatment strategies for this unfortunate group of patients.

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